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Ruptured true aneurysm of mitral valve
A complication of aortic valve endocarditis

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The case of a 32-year-old man is described who developed 'mitral regurgitation' after bacterial endocarditis on a regurgitant aortic valve. At operation this was found to be due to perforation of a large aneurysm at the base of the anterior leaflet of the mitral valve, resulting in a fistulous communication between aortic vestibule and left atrium. Repair was effected by excision of the aneurysm and mitral annuloplasty. The pathogenesis and problems of diagnosis are discussed. Accurate operative assessment of lesions of the mitral valve secondary to infective endocarditis is important as repair with preservation of normal valve mechanism may be possible.

True aneurysms of the mitral valve are uncommon and usually the result of healed bacberial endocarditis. A feature of special interest in the case to be described is that despite the large size of the aneurysm and the presence of perforation, its site of formation at the base of the anterior leaflet of the mitral valve allowed repair by excision and suture without valve replacement.

Case report

The patient was a 32-year-old Turkish civil servant with symptoms of tiredness, palpitation, and effort dyspnoea. A heart murmur was detected at the age of 10 years after an illness which was probably rheumatic fever. He remained asymptomatic until the age of 25, when he first noticed increased breathlessness on exertion. In 1966, aged 28, a diagnosis of aortic regurgitation was made and digitalis therapy started. In August 1967 he developed clinical features of infective endocarditis. A positive blood culture was not obtained, but there was a good clinical response to benzyl penicillin (1 megaunit 6 hourly for 3 weeks). During this illness his blood pressure changed from 160/70 to 150/40 mmHg. In October 1967 he was admitted to Hammersmith Hospital for assessment. A clinical diagnosis of moderate aortic and moderate mitral regurgitation was confirmed by cardiac catheterization (Table), aortography, and left ventricular cineangiocardiography. The 70 mm V wave in the pulmonary arterial wedge tracing and the normal left atrial size suggested that the mitral regurgitation was sudden and recent in onset. As his disability was only slight, valve replacement was deferred. He remained symptomatically unchanged until the end of 1969, when he began to experience increasing fatigue and exertional dyspnoea.

Physical examination on admission to Brompton Hospital in March 1970 revealed prominent arterial pulsation in the neck and a blood pressure of 140/50 mmHg. The rhythm was regular and the jugular venous pressure was not raised. The left ventricular impulse was hyperdynamic. A grade 4/6 pansystolic murmur was audible at the apex, with a third heart sound and short delayed diastolic murmur. At the left sternal border there was a grade 3/6 ejection systolic murmur and a long, quiet grade 2/6 early diastolic murmur. The lungs were clear and there was no hepatomegaly or peripheral oedema. The spleen was palpable 1 cm below the left costal margin.

Chest *x*-ray showed an enlarged heart and pulmonary venous congestion. No intracardiac calcification was seen on the image intensifier. The electrocardiogram confirmed sinus rhythm, with mean QRS axis 0°, left atrial hypertrophy, and considerable left ventricular hypertrophy (Fig. 1). The haemoglobin was 15·4 g/100 ml. The Wassermann and VDRL were negative. Cardiac catheterization was repeated, and the results are shown in the Table with those of the earlier study. The aortogram and left ventricular cineangiocardiogram showed severe aortic and mitral regurgitation.

Operation was performed on 12 March 1970, using cardiopulmonary bypass, moderate hypothermia (32°C), and perfusion of both coronary arteries. All three cusps of the aortic valve were slightly thickened and shortened by fibrosis but the main cause of regurgitation was prolapse of the non-coronary cusp below the level of the left and right coronary cusps. The appearance of the mitral valve region when viewed from the left atrium was at first confusing. This was due to the presence of an aneurysmal sac, approximately 2·5 cm in diameter and 2 cm long, arising from the base of the anterior leaflet of the mitral valve and partially obscuring it from view. The aneurysm

TABLE *Cardiac catheterization data*

Site	*October 1967*		*March 1970*	
	*Pressures (mmHg)**	*O_2 sat (%)*	*Pressures (mmHg)**	*O_2 sat (%)*
Right atrium	a=10 v=15 x= 8 y= 9	72	a=14 v=12 (11) x= 8	
Pulmonary artery	48/18 (28)	72	54/30 (38)	66
Pulmonary arterial wedge	a=22 v=70 (25) x= 9 y=18		a=27 v=47 (29) x=23 y=23	
Left ventricle	117/0–16		114/9–20	
Aorta	117/63	95	110/70	97
Cardiac output	3·3 l./min per m²		3·0 l./min per m²	

*Figures in parentheses indicate mean values. Reference level: midthorax.

was cone-shaped, with thin fibrous walls, and at its tip there was a 1 cm diameter perforation (Fig. 2). When the aortic vestibule was re-examined after the aortic cusps had been excised, the origin of the aneurysm could be clearly seen about 1·5 cm below the posterior third of the noncoronary cusp (i.e. below that third adjacent to the commissure between noncoronary and left coronary cusps). The perforated aneurysm thus represented an intracardiac fistula between the outflow tract of the left ventricle and the left atrium, giving rise to a haemodynamic situation indistinguishable from mitral regurgitation. The aneurysm was excised at its neck and the resulting defect between left atrium and left ventricle closed by direct suture. The remainder of the anterior leaflet of the mitral valve, the posterior leaflet, and the chordae tendineae appeared normal. As the mitral annulus was moderately dilated an annuloplasty was performed and this restored competence of the valve. A Starr-Edwards prosthesis was sutured into the aortic annulus. After operation the patient made an excellent recovery and at the time of discharge there was no evidence of valvar regurgitation or intracardiac shunt.

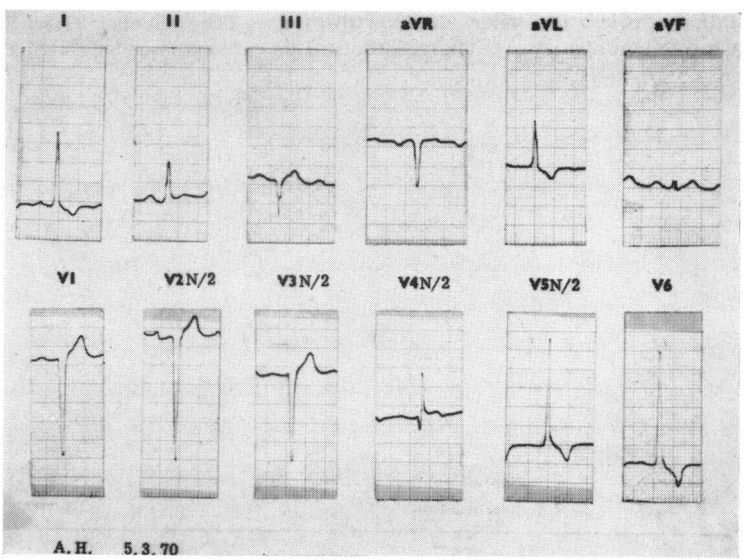
Portions of the aneurysm were examined histologically. There was no endothelial lining on either ventricular or atrial aspect of the aneurysm. The base of the aneurysm was composed of fibrous tissue with elastic fibres. The latter rapidly became attenuated, and were absent from the wall of the aneurysm near the site of perforation. No inflammatory cells were seen.

Discussion

Aortic regurgitation complicated by infective endocarditis may result in retrograde dissemination of bacteria and secondary involvement of the mitral valve (Edwards and Burchell, 1958). Chordal rupture, mycotic false aneurysm, or, more rarely, true aneurysmal dilatation of a valve leaflet with or without perforation may follow. Aneurysms, when present, are usually found near the base of the anterior cusp (Hudson, 1965).

Saphir and Leroy (1948) stressed the rarity of true aneurysms of the mitral valve in the era before penicillin was available for the treatment of subacute bacterial endocarditis. Maclean and MacDonald (1957) confirmed this finding, and observed that large well-organized aneurysms with smooth fibrous walls could only develop if the disease was sufficiently prolonged to allow healing to accompany the infective process. Under such circumstances the young scar tissue present in an area of circumscribed valvulitis may yield to intracardiac pressure and form a true aneurysm. More recently, Gonzalez-Lavin, Lise, and Ross (1970) have reviewed the surgical implications of infective endocarditis

FIG. 1 *The electrocardiogram showing sinus rhythm, mean QRS axis 0°, left ventricular hypertrophy with T wave inversion in leads I, aVL, V4, V5, and V6.*



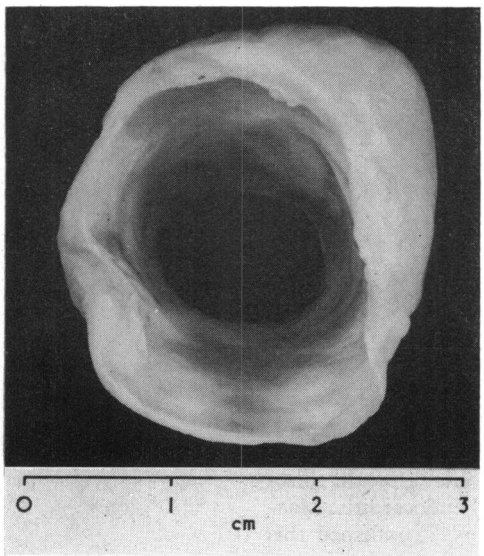


FIG. 2 *Photograph of excised aneurysm viewed from the left ventricular aspect. The large perforation at its tip is clearly visible.*

involving the left side of the heart. Secondary involvement of the mitral valve was present in 10 out of 58 patients operated upon for aortic regurgitation due to bacterial endocarditis. True aneurysm formation on the anterior leaflet of the mitral valve, but without perforation, occurred in 2 of these.

The present case illustrates the difficulty in diagnosing these aneurysms correctly, even after rupture. The haemodynamic findings at cardiac catheterization and angiocardiography do not distinguish this situation from regurgitation through the valve orifice. However, an antecedent history of infective endocarditis on pre-existing aortic regurgitation should suggest that the mitral valve may have become secondarily affected by chordal rupture, cusp perforation, or aneurysm formation.

At operation it is important to determine the precise pathological anatomy, because, depending on the nature and site of the mitral lesion, conservation of the mitral valve may be possible. A repair operation may be possible in some cases of ruptured chordae tendineae (McGoon, 1960), or, as in the patient described, an aneurysm may be excised with preservation of normal valve mechanism.

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